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OUTBREAK OF MUMPS AMONG YOUNG ADULTS — VANCOUVER, BRITISH COLUMBIA

Mumps is an acute viral disease, caused by the infectious agent *Paramyxovirus*. It is defined for surveillance purposes as an illness characterized by fever and tender, self-limited swelling of the salivary gland(s), lasting ≥ 2 days without other apparent cause[1]. In January and February 1997, an unexpected number of cases of mumps were reported to the Vancouver/Richmond Health Board (V/RHB). An investigation was initiated with the British Columbia Centre for Disease Control (BCCDC). Between 1 January 1997 and 31 March 1997, a total of 83 cases of mumps were reported via the BC Communicable Disease Surveillance System.

Figure 1 shows the epidemic curves for Vancouver and the rest of BC. The V/RHB notified Vancouver physicians of the mumps outbreak during the week of 26 February to 4 March, and a press release with public information about the outbreak was issued on 3 March.

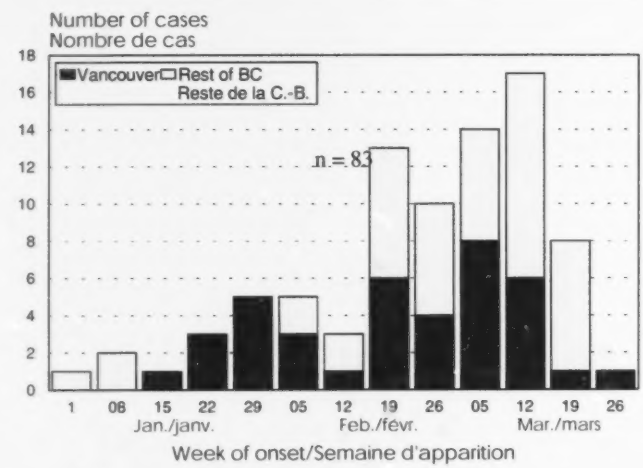
Forty-six percent (38/83) of the cases were within the Vancouver area of the V/RHB. The rate of mumps in BC increased from the 1992 to 1996 baseline of 1.4 per 100,000 person years to 8.4 per 100,000 person years in January to March 1997 (rate ratio = 6.2); the corresponding increase in the Vancouver area was from 0.2 to 27.8 (rate ratio = 126). Fifty-one percent (41/81) of the outbreak cases for whom gender was known were female; gender was not known for two cases. Fifty-five percent (46/83) were 15 to 24 years old and the median age was 20 years.

Table 1 shows the rate (per 100,000 person years) for mumps reported from 1992 to 1997 by birth cohorts. Persons born from 1971 to 1980 (17 to 26 years old) experienced the highest rate of mumps during this outbreak (33.5 per 100,000 person years). During the outbreak period, the rates in the birth cohorts 1971 to 1980 and 1957 to 1970 both increased more than 18 times the baseline rate.

A universal combined measles, mumps, and rubella (MMR) immunization program was introduced in BC in 1981 for children aged ≥ 12 months.

Initial case follow-ups by the V/RHB suggested that public social events were risk factors for acquiring mumps in the young

Figure 1
Epidemic curve for mumps in British Columbia and Vancouver, 1 January to 31 March 1997*



* If onset date was unavailable, date of report was used.

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adult age group. A case-control study was initiated to examine this hypothesis and to further explore the outcomes of mumps illness.

Cases were selected from those reported to the surveillance system by 21 March 1997. Fifty-one cases were administered a standard questionnaire by telephone interview; the identified regular physicians were asked to select age and gender matched controls from their patient lists. The physicians of 15 cases declined to participate; for these cases matched controls were obtained from physician lists from the same geographic area. Controls who reported a past history of mumps were excluded. Forty-seven matched case-control pairs were analyzed.

Twenty-two percent (11/51) of the cases reported physician-diagnosed extra-salivary manifestations of mumps, including pancreatitis, thyroiditis, and orchitis. Thirty-three percent of the male cases > 16 years old (7/21) reported testicular pain during the 2 weeks following salivary swelling. Eleven percent (6/51) of all cases reported upper abdominal pain and 47% (24/51) reported severe headache during these 2 weeks.

Vaccine effectiveness, ascertained by self-reported immunization status, was estimated to be 80% (95% confidence interval [CI] = 29% to 96%).

Eighty-eight percent (45/51) of the cases reported being in the Greater Vancouver area during the exposure period. Among the 47 matched case-control pairs, contact with a person with mumps was significantly associated with illness (odds ratio = 13, 95% CI = 1.95 to 552). Sixteen of the 40 cases aged 17 to 40 years, and one of the nine cases aged < 17 years reported attending a "rave" party during the exposure period. "Raves" are very large gatherings of teenagers and young adults who listen and dance to electronic music in warehouse type venues. Of the risk factors examined, exposure to rave parties was the most strongly associated with illness (odds ratio = 17, 95% CI = 2.7 to 710). All "rave" attendees reported sharing drinks and dancing in close proximity to others. Attending educational institutions, having contact with > 50 persons per day at work, being a health-care professional, attending dance clubs, bars, or other social events, and playing group sports were not associated with illness.

The increased incidence rates in this outbreak suggest that the 1957 to 1980 birth cohorts were most at risk. These incompletely vaccinated birth cohorts sustain an adult population susceptible to outbreaks of mumps in the future. This investigation highlights, as previously described[2,3], the high proportion of extra-salivary symptoms of mumps experienced by an adult population.

This is the second communicable disease outbreak reported to be associated with "rave" parties in Canada, the first being a 1996 meningitis outbreak in Quebec[4]. These social events, characterized by close contact and sharing of drinks, should be considered as a risk factor for mumps and other illnesses transmissible by person-to-person droplet spread.

In response to the outbreak and this investigation, the V/RHB released a public notice about "raves" as a risk factor for acquiring mumps and a reminder about MMR vaccinations.

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Source: *A Bell, MDCM, MHSc, M Fyfe, MD, MSc, Communicable Disease Epidemiology Services, BCCDC, M Bigham, MD, MHSc, P Daly, MD, V/RHB, J Buxton, MBBS, MHSc, Community Residency Program, Department of Health Care and Epidemiology, UBC, Vancouver, BC; C Craig, DVM, BSc, Field Epidemiology Training Program, LCDC, Ottawa, ON.*

Table 1
Rates of mumps incidence in British Columbia, by birth cohort groups

Birth cohort	Age in 1997 (years)	Vaccination program available	Rate per 100,000 person years (1992-1996)	Rate per 100,000 person years (Jan-Mar 1997)	Rate Ratio
before 1956 - 1956	40+	None	0.15	0.74	5.0
1957-1970	27-40	None	0.36	6.51	18.1
1971-1980	17-26	None	1.74	33.49	19.2
1981-1990	7-16	Infant MMR	4.95	7.04	1.4
1991 - after 1991	0-7	Infant MMR	0.92	4.59	5.0

FATAL HUMAN PLAGUE — ARIZONA AND COLORADO, 1996

In 1996, five cases of human plague, of which two were fatal, were reported in the United States; both decedents had septicemic plague that was not diagnosed until after they died. This report summarizes the investigation of the two fatal cases and underscores the need for health-care providers in areas with endemic plague to maintain a high level of awareness about the risk for plague in their patients.

Patient 1

On 2 August, 1996, an 18-year-old resident of Flagstaff, Arizona, was taken to a local outpatient clinic because of a 2-day history of fever, pain in his left groin, and diarrhea. On examination, he was afebrile, had a pulse rate of 126 beats per minute, respiration rate of 20 breaths per minute, and blood pressure of 130/81 mm Hg. Left groin swelling and tenderness were noted. A groin muscle strain was diagnosed and attributed to a fall 2 days earlier. He was treated with nonsteroidal anti-inflammatory agents, instructed about using a liquid diet, and released. On 3 August, the patient reported feeling weak, had difficulty breathing, and collapsed while taking a shower. Emergency medical assistance was called, and the patient experienced cardiac arrest while emergency medical technicians were on site. He was transported to a hospital emergency department (ED) and pronounced dead shortly after arrival.

On 8 August, cultures of blood samples obtained in the ED were presumptively positive for *Yersinia pestis* by fluorescent antibody staining and confirmed by specific bacteriophage lysis at the laboratory of the Arizona State Department of Health. Additional isolates from postmortem brain, liver, lung, and vitreous fluid cultures were confirmed as *Y. pestis* at the Centers for Disease Control and Prevention (CDC). An epidemiologic investigation by public-health officials indicated that the patient most likely became infected on 27 July as the result of bites by *Y. pestis*-infected fleas while walking through a prairie dog *(Cynomys gunnisoni)* colony in Navajo County. High antibody titers to the fraction 1 (F1) antigen of *Y. pestis* were detected in two of four pet dogs living in houses near the prairie dog colony. Dog owners were advised about the risk for plague and instructed to restrain their pets and to periodically dust them with insecticide. Prairie dog burrows within one-half mile of the residences were dusted with insecticide to control flea populations.

Patient 2

On 17 August, 1996, a 16-year-old resident of western Colorado had onset of pain followed by numbness in her left arm and left axillary pain. During 18 and 19 August, she had chills, fever, and several episodes of vomiting. On 19 August, she was evaluated at a local hospital ED. Findings included a temperature of 97.4° F (36.3° C), pulse rate of 100 beats per minute, respiration rate of 16 breaths per minute, and blood pressure of 103/59 mm Hg; a chest radiograph was interpreted as within normal limits. She was discharged with a diagnosis of possible brachial plexus injury related to a fall from a trampoline on 14 August. She was prescribed analgesics, and an appointment with a neurologist was scheduled.

On 21 August, she was found semiconscious at home and taken to the same hospital. She was confused and complained of neck pain and generalized soreness. Findings on examination included a temperature of 102.5° F (39.2° C), pulse rate of 170 beats per minute, respiration rate of 50 breaths per minute, and blood pressure of 130/70 mm Hg. Within an hour of arrival at the hospital, she experienced respiratory arrest and was intubated. Numerous gram-positive diplococci were detected in a blood smear, and a chest radiograph revealed bilateral pulmonary edema. She was administered 2 g ceftriaxone intravenously and transferred to a referral hospital with diagnoses of septicemia, disseminated intravascular coagulation, adult respiratory distress syndrome, and possible meningitis. A gram stain of sputum revealed rare white blood cells and no bacteria; she was treated for gram-positive sepsis. However, her condition rapidly deteriorated, and she died later that day.

On 23 August, blood and spinal fluid cultures obtained on 21 August grew an unidentified gram-negative rod and *Streptococcus pneumoniae*. On 26 August, *Yersinia pseudotuberculosis* was initially identified in cultures of blood and respiratory aspirate using a rapid microbiologic identification device. This blood culture isolate subsequently was presumptively identified as *Y. pestis* at the Utah Division of Laboratory Services and confirmed as *Y. pestis* at CDC.

An environmental investigation by health officials revealed evidence of an earlier extensive prairie dog die-off adjacent to the patient's residence. High antibody titres to the F1 antigen of *Y. pestis* were present in serum specimens from four of five family dogs and one of three family cats. The seropositive cat had a submandibular lesion consistent with a healing abscess. Family members reported that the cat had been recently ill and had been closely cared for by the decedent. Investigators concluded that the decedent was probably exposed to *Y. pestis* by direct contact with infectious material while handling the cat. None of 10 flea pools or 13 rodents (least chipmunk, *Tamias minimus* [four]; deer mouse, *Peromyscus maniculatus* [six]; and house mouse, *Mus musculus* [three] collected on the property tested positive for *Y. pestis* or for antibody to *Y. pestis*, respectively. Because the diagnosis was established after the standard 7-day maximum plague incubation period had elapsed, antibiotic prophylaxis of family members and medical personnel was not instituted.

MMWR Editorial Note

In the United States, most cases of human plague are reported from New Mexico, Arizona, Colorado, and California[1,2]. The principal forms of plague are bubonic, septicemic (primary and secondary), and pneumonic (primary or secondary). From 1947 through 1996, a total of 390 cases of plague were reported, resulting in 60 (15.4%) deaths. Of these, bubonic plague accounted for 327 (83.9%) cases and 44 (13.5%) deaths; primary septicemic plague, for 49 (12.6%) cases and 11 (22.4%) deaths; and primary pneumonic plague, for seven (1.8%) cases and four (57.1%) deaths. Seven (1.8%) cases were unclassified, including one (14.3%) death (CDC, unpublished data, 1997). During 1965-1989, a total of 27 persons with plague were treated at the Gallup Indian Medical Center in New Mexico. Of these, classic signs of bubonic plague were present in only 10 (37%); provisional diagnoses in other patients included apparent upper respiratory tract infections, nonspecific febrile syndromes, gastrointestinal or urinary tract infections, or meningitis[3]. The syndromes in both patients

described in this report initially were attributed to injuries and treated with analgesics.

Bubonic plague may not be considered by a physician if swollen, tender lymph nodes are not detected or present on physical examination. Evidence of regional lymphadenitis should prompt a suspicion of plague in a patient who lives in or has recently visited an area with endemic plague. Septicemic plague without obvious lymphadenopathy is more difficult to diagnose because the manifestations are non-specific (e.g. elevated temperature, chills, abdominal pain, nausea, vomiting, diarrhea, tachycardia, tachypnea, and hypotension)[4].

A patient with clinical signs of sepsis and a history of possible plague exposure, particularly during the spring, summer, and fall months, should be aggresively managed as having plague. Even before a specific laboratory diagnosis is obtained, antibiotic therapy should be initiated with streptomycin; alternatives include gentamicin, chloramphenicol, and the tetracyclines. The penicillins and cephalosporins are not effective in treating plague, although these drugs frequently show activity in vitro[5].

In suspected cases of plague, several samples of blood should be collected for culture during a 45-minute period before initiation of antibiotic treatment, unless such a delay is contraindicated by the patient's condition. The direct immunofluorescence test for the rapid presumptive identification of *Y. pestis* F1 antigen should be applied to appropriate clinical materials (e.g. lymph node aspirates, culture isolates, or blood films), and if pneumonic plague is suspected, tracheal washes or sputum smears. Rapid microbiologic identification devices may not include adequate *Y. pestis* profiles in their database and, therefore, may misidentify *Y. pestis* as *Y. pseudotuberculosis*[6]. Acute- and convalescent-phase serum specimens should be obtained to detect antibodies to the *Y. pestis* F1 antigen by using passive hemagglutination assay or enzyme-linked immunosorbent assay methods.

Control measures to prevent human plague include surveillance for plague in rodents and rodent predators as well as public education.

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Source: *Morbidity and Mortality Weekly Report, Vol 46, No 27, 1997.*

Erratum

CHLAMYDIA TRACHOMATIS IN CANADA: AN UPDATE
Vol. 23-15, page 114

The second sentence in the second paragraph on page 114, "An estimated 40% of men and 60% of women with chlamydial infections have concurrent infections with gonorrhea," is incorrect. The sentence should read as follows: "An estimated 40% of men and 60% of women **with gonorrhea** have concurrent infections **with *Chlamydia***."

The proportions of male and female patients with gonorrhea who are co-infected with *Chlamydia* are based on the evidence derived from a review of the scientific literature. A summary of recent research findings on co-infection is listed below.

- Approximately 15% to 25% of men and 30% to 50% of females with acute gonorrhea are co-infected with *Chlamydia*[1].
- Co-infection with *Chlamydia* occurs in 15% to 25% of men and 30% to 40% of females with gonorrhea[2].
- Co-infection with *Chlamydia* is common (20% to 30% of symptomatic men and 30% to 50% of women with gonorrhea)[3].
- An estimated 44% to 79% of women with gonorrhea also have an infection with *C. trachomatis*[4].
- 56.5% of pregnant teenagers with gonorrhea were co-infected with *Chlamydia*[5].
- 20% of men and 38% of women with gonorrhea had concurrent *Chlamydia*[6].
- 44% of female adolescents with gonorrhea were co-infected with *Chlamydia*[7].

In the literature cited above, co-infection is reported as a proportion of all patients infected with gonorrhea. In other words, the denominator is the total number of gonococcal infections (including those with co-infections).

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